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IN THIS ISSUE

Negro Mortality, Trends, 1920-44

Q Fever Studies in Southern California
Two New Salmonella Types



CONTENTS

Negro mortality, III. Course of mortality from specific causes, 1920-4 Mary Gover
Deaths during week ended January 17, 1948
Q fever studies in Southern California, I. Recovery of Rickettsia burne
from raw milk. R. J. Huebner, W. L. Jellison, M. D. Beck, R. I
Parker and C. C. Shepard
Two new Salmonella types. James Watt, Thelma M. DeCapito, P. I
Edwards and Alice B. Moran
Edwards and Ance B. Moran
INCIDENCE OF DISEASE
United States:
Reports from States for week ended January 24, 1948, and comparison
with former years
Weekly reports from cities:
City reports for week ended January 17, 1948
Rates, by geographic divisions, for a group of selected cities
Foreign reports:
Canada-Provinces-Communicable diseases-Week ended Janu
ary 3, 1948
Reports of cholera, plague, smallpox, typhus fever, and yellow fever
received during the current week-
Smallpox
Yellow fever

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NEGRO MORTALITY

III. COURSE OF MORTALITY FROM SPECIFIC CAUSES, 1920-1944 1

By MARY GOVER, Biostatistician, United States Public Health Service

Page

201

213

214

223

225

229

231

232

232 232 The course of mortality from specific causes is of great importance in considering the general aspects of Negro mortality in the United States. In presenting the trend of mortality it is well to control as many of the major factors affecting mortality as practical. Age is of prime importance in this connection; sex is relatively less so since males and females are usually about equally represented in a population; a constant area is advisable. However, the area of the expanding death registration States includes the entire country from 1933 on, and for every year it comprises all of the Negro population living in States where registration of deaths met the requirements set by the Bureau of the Census.

Differences in the age distribution of nonwhite and white populations are clearly marked in any enumeration, the nonwhite population having proportionately more children and fewer old people than the white. In the 1940 Census, among nonwhite and white, respectively, 9.8 and 7.8 percent of the population were children under 5 years of age and 4.8 and 7.1 percent were persons 65 years of age or older. Not only do the age distributions of nonwhite and white populations differ at a specific time but there has been a gradual aging of the population evident in successive enumerations. The percentage of persons 65 years and over in the 1920, 1930, and 1940 enumerations was 3.2, 3.2, and 4.8 for nonwhite and 4.8, 5.7, and 7.1 percent for white, respectively.

Adjustment of mortality rates at all ages for differences in age composition of the population for comparative purposes has been made by the direct method of adjustment, that is, by applying observed age-specific rates to a population chosen as standard.² Since (1) non-white and white rates are adjusted to the same standard population,

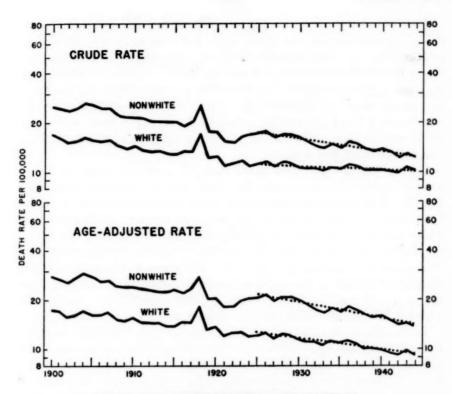
¹ From the Division of Public Health Methods. This is the third in a series of studies of Negro mortality (1) consisting of data assembled principally from reports of the U. S. Bureau of the Census and prepared at the request of the Office of Negro Health Work, U. S. Public Health Service.

² For further details of the method see Raymond Pearl: Medical Biometry and Statistics, 3rd edition, pp. 274-276.

namely, the total enumerated population of the United States in 1940. and since (2) the enumerated nonwhite population is relatively younger than the white, it follows that age adjustment of the crude rates frequently raises the nonwhite rate by a considerable amount, and on the other hand makes a comparatively small change in the white rate. This is the case when crude nonwhite and white rates for 1940 are adjusted to the total population in 1940 with the result that nonwhite rates are higher and white are lower than before adjustment for age has been made. In the present study of trends of mortality a further purpose of age-adjustment of crude rates is to make a comparison of successive annual rates of nonwhite and white mortality uninfluenced by age changes in the populations. Such age changes in the past have been more rapid for white than nonwhite elements of our population. The rate of decrease of age-adjusted rates for all causes can be compared also, since adjustment has been made to the same standard population for both nonwhite and white mortality.

In the charts which follow, annual rates for those causes of death which have a high mortality in old age have been adjusted for age (figs. 3 and 4); the remainder (figs. 2, 5, 6, and 7) are constructed from crude rates. In adjusting for age the following method was used: rates specific for cause and color for the years 1920-21, 1929-31, 1939-41, 1941, 1942, and 1943 were adjusted for age in the usual way, using the enumerated population of the United States in 1940 as the The ratio of the adjusted to the crude rate was computed for the same years, interpolations between these ratios giving annual ratios for successive intervening years. The annual ratios were then applied to the crude rates to obtain an age-adjusted rate for each year (see footnote 8, table 2). Since age-specific populations must be obtained by interpolation between census enumerations in order to adjust each annual rate for age by the direct method, it seems that the shorter method of interpolating the ratio and applying it to the crude rate for each year is accurate enough.

Figure 1 shows the rate of decline of nonwhite and white mortality from all causes for both crude and age-adjusted rates in the expanding area of the death registration States, from 1900 to 1944. Prior to 1925 the death registration States included a relatively small proportion of the total nonwhite population; 5 percent in 1900, 12 percent in 1910, 66 percent in 1920, and 86 percent in 1930. For this reason the period 1925-44 was chosen for the construction of trend lines. Straight lines have been fitted by the method of least squares to the logarithms of the nonwhite and white rates, both crude and adjusted, for 1925 to 1944. The average annual percentage decline of the fitted lines and the probable error of the decline is shown in figure 1; namely, crude nonwhite, -1.77 ± 0.095 ; crude white, -0.49 ± 0.075 ; age-



	DEATH	RATE PER	100,000	
	CRU	DE	ADJU	STED
YEAR	NON WHITE	WHITE	NON WHITE	WHITE
1919-1921	17.0	12.0	19.8	13.1
1929-1931	16.2	10.9	20.0	11.8
1939-1941	13.5	10.3	15.9	10.0
AV	ERAGE ANN	UAL PERCE	NTAGE CHA	NGE
1930 to 1940 laverage rates	-1.7	6	-2.1	-1.5
1925 to 1944 (fitted line)	-1.77 ±.095	49 2.075	-2.08 2.099	-1.51 ± . 090

FIGURE 1.—Crude and age-adjusted mortality from all causes, nonwhite and white persons in the death registration States, 1900-1944. Trend lines, represented by dotted lines, have been fitted to the logarithms of the rates, 1925-1944.

adjusted nonwhite, -2.08 ± 0.099 ; and age-adjusted white, -1.51 ± 0.090 .

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where Y is the logarithm of the observed rate

b=(antilog of B)-1 where b is the average annual percentage change in the rates

$$\sigma_B = \frac{\overline{\sigma}}{\sqrt{\Sigma (\mathbf{x} - \bar{\mathbf{x}})^3}},$$

where $\overline{\sigma}$ is the square root of the sum of the squares of the differences between the observed and calculated values of Y (logarithms) divided by n-2, in which n is the number of years (20) used in fitting the straight line, and $\Sigma(x-\hat{x})^2$ is the sum of the squares of the deviations of x from the mean of x.

 $PE_b = 0.67449 \frac{(1+b)\sigma_B}{0.4342945}$

³ The average annual percentage decline and probable error was computed from straight lines fitted to the logarithms of annual rates as follows:

The rate of decline in mortality during the last 20 years is increased by age-adjustment of the crude rates for both nonwhite and white persons (fig. 1). The change in the annual rate of decline brought about by adjustment for age is not significant for the nonwhite, that is, from -1.77 ± 0.095 to -2.08 ± 0.099 percent; while for the white the change from -0.49 ± 0.075 to -1.51 ± 0.090 percent is statistically significant. That is, age-adjustment of mortality from all causes increases the rate of decline in the rates only slightly for nonwhite and significantly so for white.

During the last 20 years crude rates of mortality from all causes have been declining at a somewhat faster rate for nonwhite than white (fig. 1). Rates adjusted for age also show a more rapid rate of decline for nonwhite. The difference between the rates of decline for nonwhite and white is less for age-adjusted than for crude rates, owing to the greater acceleration in the rate of decline of the white rates occasioned by age-adjustment. The difference between the rate of decline (1925–44) in age-adjusted nonwhite mortality, -2.08 ± 0.099 percent, and that for white, -1.51 ± 0.090 percent, is -0.57 ± 0.134

percent, or a small but significant difference.

In the charts (figs. 2-7) which follow, showing the trend of mortality from specific causes, the rates of decline are predominantly faster among whites, in spite of the more rapid rate of decline in mortality from all causes among the nonwhite. The chief reason for the apparent discrepancy is the faster rate of decline in the relatively large rate for ill-defined causes among the nonwhite. Other contributing factors are the absence of any marked increase in heart disease mortality among nonwhite, and the fact that tuberculosis, which is declining rapidly, is a relatively larger part of the total death rate among nonwhite.

Figures 2–7 show the course of mortality from selected causes among nonwhite and white and are self-explanatory. The rates are for the expanding death registration States and have been adjusted for age for the causes shown in figures 3 and 4; crude rates are shown in figures 2 and 5–7. Average rates for three successive decades are tabled opposite the charts for each specific cause together with the average annual increase or decrease in the rates from 1930 to 1940.

Communicable diseases (fig. 2) including tuberculosis (fig. 3) have decreased rapidly in recent years. The slower rate of decline for the nonwhite may be associated with a lower rate of immunization and less extensive use of the sulfa-compounds. Pneumonia (fig. 4), pellagra (fig. 5) and malaria (fig. 5) have also shown spectacular rates of decline. Syphilis (fig. 4) has been declining in very recent years among nonwhite.

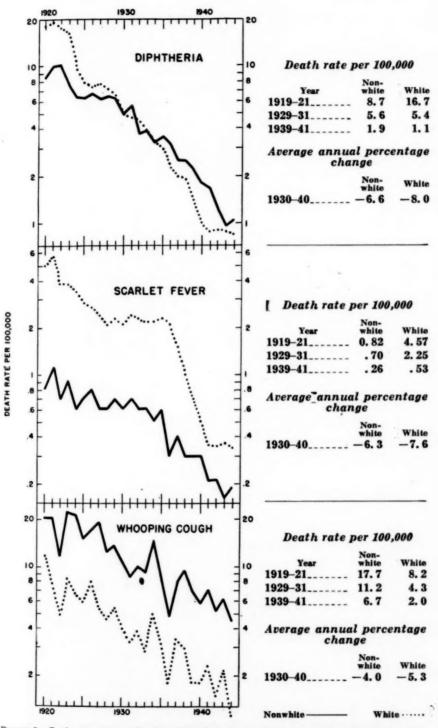


FIGURE 2.—Crude rates of mortality from selected causes—nonwhite and white mortality in the death registration States, 1920-1944.

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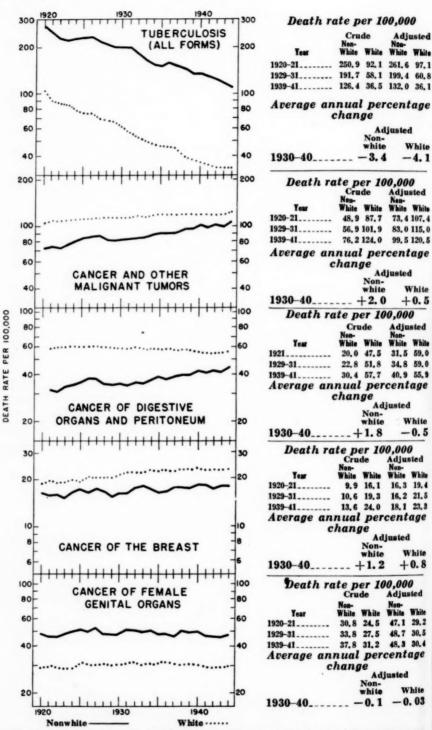


FIGURE 3.—Age-adjusted rates of mortality from selected causes—nonwhite and white mortality in the death registration States, 1920-1944.

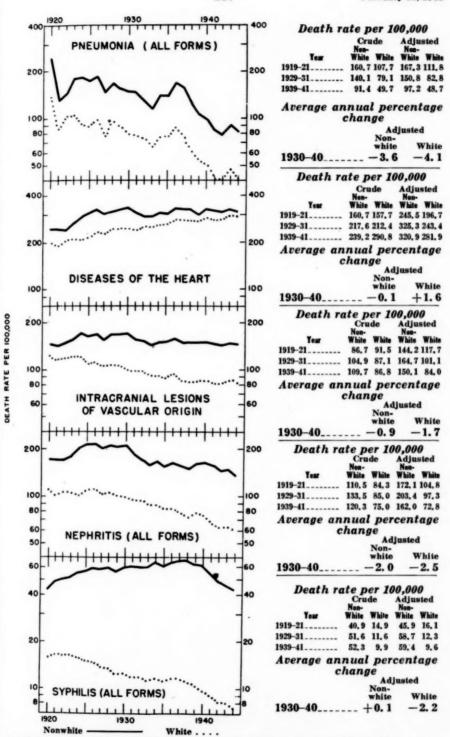


Figure 4.—Age-adjusted rates of mortality from selected causes—nonwhite and white mortality in the death registration States, 1920-1944.

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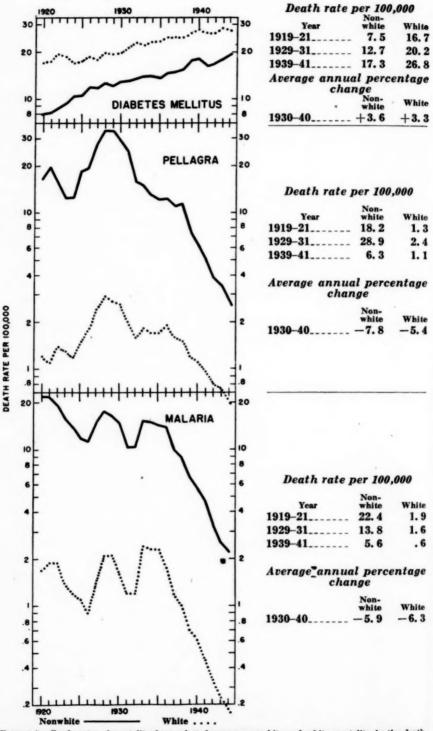


Figure 5.—Crude rates of mortality from selected causes—nonwhite and white mortality in the death registration States, 1920-1944.

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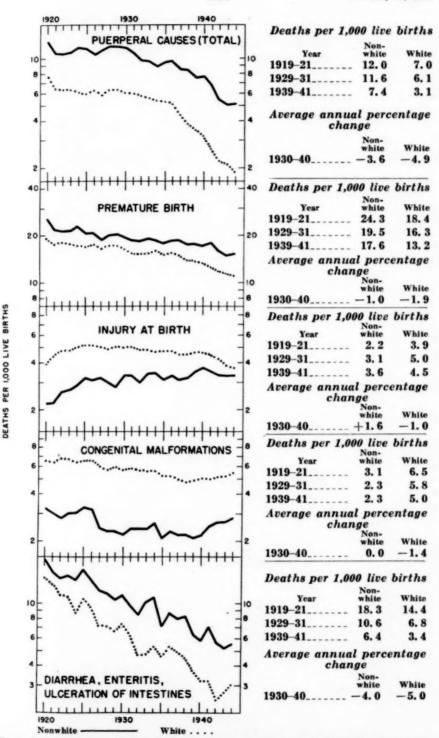


Figure 6.—Mortality from selected causes, deaths under 1 year of age, per 1,000 live births and maternal mortality—nonwhite and white mortality in the death registration States, 1920-1944.

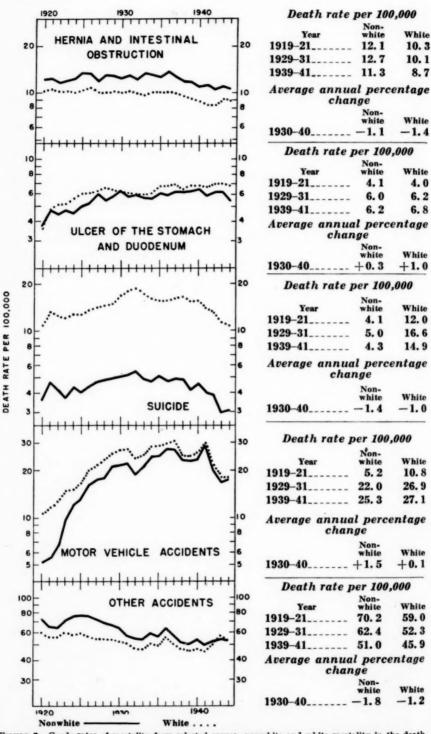


Figure 7.—Crude rates of mortality from selected causes—nonwhite and white mortality in the death registration States, 1920-1944.

Table 1.—Mortality from selected causes, 1941-45

Cause of death			Nonwhite					White		
Transport of Great	1941	1942	1943	1944	1945	1941	1942	1943	1944	1945
				R	Rate per 100,000 population	00 population				
Age-adjusted 1	1, 324.91	1, 245.62	1, 276.45	1, 240. 47	1, 204, 65	1, 011. 05 972. 46	1,003.57	1,067.67	1,044.15	1,044.90
				Crud	Crude rate per 100,000 population	0,000 populat	ion			
Diphtheria Scarlet fever Whooping cough Tuberculosis (all forms) Cancer and other malignant tumors Cancer of female genital organs at Cancer of female genital organs Cancer of female genital organs Cancer of the heast Cancer of the heast Cancer of the heart Cancer of the heart Intracranial esions of vascular origin Nephritis (all forms) Diabetes mellitus Pellagra Malaria Malaria Malaria Mulcor-vehicle accidents Other accidents	12.22 12.22 12.23 12.25	1. 2. 2. 2. 2. 2. 2. 2. 2. 2. 2. 2. 2. 2.	0 6 25 25 25 25 25 25 25 25 25 25 25 25 25	1. 4814888484848484888888888888888888888	1.38 1.00 1.00 1.00 1.00 1.00 1.00 1.00 1.0	0 ,4842 242 252 252 252 252 252 252 252 252 2	6.17.48.88.88.88.88.88.88.88.88.88.88.88.88.	0. 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2 2	0 1. 1883 1. 288 2. 288	21.1. 138. 23.0 68. 23.0 69. 23. 24. 25. 25. 25. 25. 25. 25. 25. 25. 25. 25
					Rate per 1,000 live births	live births				
Puerperal causes (total) Premature birth Injury at birth Congenital malformations Diarrhea, enteritis, ulceration of intestines.	6.78 17.90 3.51 7.07	5.44 15.97 3.29 5.59	14.98 14.98 2.28 5.17	55.50 52.50 52.50 53.50 53.50 54.50 55.50	4.8.8.4. 3.11.8.4. 7.6.4.97	12.26 25.45	11.81 11.81 4.18 2.15 2.42	11.36 2.38 2.22 2.22 2.00	11.37 11.37 3.70 3.40 3.00	11.72 11.09 3.63 5.92 2.73

1 For crude death rates in the death registration States, 1900-1940, see reference (2). The see-adjusted to the total population as numerated in 1940.

* For female population.

TABLE 2.—Mortality from selected causes, adjusted for age, 1920-44

ms) ?	White		は、 は、 は、 は、 は、 は、 は、 は、 は、 は、
Syphilis (all forms) 7	Non- white		చిశ్రాలో మైద్ది స్టేష్ట్రి స్టేష్ట్రి ప్రాప్తి ప్రస్తి ప్రాప్తి ప్రస్తి ప్రాప్తి ప్రస్తి ప
ritis ms) 6	White		100.1011 100
Nephritis (all forms) ⁶	Non- white		173.1 171.0 172.7 173.7 179.7 179.7 179.7 179.8 199.8
anial ns of ular in 4	White		120. 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8 8
Intracranial lesions of vascular origin *	Non- white		1446.6 1466.6
ses of	White		199, 8 1193, 5 2 121, 1199, 11
Diseases of the heart 4	Non- white	age 8	245.8 2845.3 2807.1 2802.1 2802.1 2802.1 2802.1 2802.2 2805.2 2805.2 2805.2 2805.2 2805.2 2805.2 2805.2 2805.2
nonia rms)	White	Rate per 100, 000 population, adjusted for age \$	136 101 101 101 101 101 101 101 10
Pneumonia (all forms)	Non- white	on, adju	204 1129 129 129 129 129 129 129 12
er of gen- gans	White	populati	0.00 0.00 0.00 0.00 0.00 0.00 0.00 0.0
Cancer of female gen- ital organs	Non- white	100,000	\$\frac{1}{2}\frac{1}\frac{1}{2}\f
east 3	White	tate per	00000000000000000000000000000000000000
Cancer of the breast 3	Non- white		15.00
er of trive s and seum ?	White		6.000 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
Cancer of digestive organs and peritoneum	Non- white		3.3.3.3.3.3.3.3.3.3.3.3.3.3.3.3.3.3.3.
r and nalig- imors	White		100.00
Cancer and other malig- nant tumors	Non- white		72.2 74.4 74.4 74.4 74.4 74.4 74.4 74.4
rms)	White		4 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0 0
Tuberculosis (all forms)	Non- white		273.7 228.5 228.2 228.2 227.7 227.7 227.7 220.0 207.6 207.6 207.6 207.6 207.6 207.7
Year		L	1920 1921 1923 1925 1925 1925 1926 1926 1930 1931 1931 1931 1931 1931 1931 1931

For purposes of comparability, the Bureau of the Census has pointed out changes in classification, or has made transfers of deaths in some cases, which are fully described in the notes to the tables in the above reference. Some of these notes are included among the notes appended to this table.

Mexicans are highles in the white in all populations. Deaths of Mexicans were recorded as colored in 1830-38, 500 and 1831 they have been tabulated and transferred to 1 For crude death rates in the death registration States, 1900-1940 see reference (2).

white deaths; for 1932-34 they were included with the nonwhite; in all other years deaths of Mexicans are tabulated as white deaths.

⁵ Includes all embolism and thrombosis, except puerperal, in 1920. ³ Per female population. Excludes cancer of the pancreas in 1920.

Excludes disease of coronary arteries, 1920–29. See also footnote 6 to this table.

**Certain terms relating to combined cardiorenal conditions were transferred from diseases of the heart to nephritis in 1939 and following. 7 Includes the total of "aneurysm" (except of heart)" for 1921–38; includes only aneurysm of the aorta for 1939 and following.

Death rates specific for cause and color for 1926-21, 1939-41, 1941, 1942, and 1943 were adjusted for age by the direct method to the total population of the United States as enumerated in the total population of the standard. A ratio or adjusted the total population of the standard. A ratio or adjusted the total population of the standard is also or adjusted to the total population of the standard in a ratio or adjusted to the standard the standard in the policy of the ratios were made between the ratios for successive decades (1920-30 and 1930-40). Annual rates adjusted for age were then obtained by multiplying the crude rate by the ratio of the adjusted to the crude. The adjusted rate for 1944 was obtained by multiplying the crude rate for 1944 by the ratio obtained for 1943. Rates shown on the charts are averages of the crude annual rates as computed by the Bureau of the Census (2), and averages of the annual rates adjusted for age as described above. Cancer (fig. 3), heart disease (fig. 4), and diabetes (fig. 5) are among the causes which have increased markedly in recent years. Cancer of the digestive organs and peritoneum has shown a marked increase in the rate among nonwhite. Heart disease has been increasing among white but shows no increase in the age-adjusted rates for non-white, in recent years.

Causes of death peculiar to early infancy are computed per 1,000 live births; among the causes shown (fig. 6) mortality from injury at birth has increased among the nonwhite; mortality in the first year of life from congenital malformations has increased since 1940 among both nonwhite and white. Maternal mortality and premature births have decreased more rapidly since 1935 among white than nonwhite.

Table 1 shows mortality rates (not adjusted for age) for the specific causes shown in figures 2-7 for the years 1941-45; rates for the years 1920-40 can be obtained from vital statistics rates in the United States 1900-40 (2). Table 2 shows mortality rates adjusted for age, as described earlier, for the causes of death shown in figures 3 and 4.

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- Gover, Mary: Negro Mortality. I. Mortality from all causes in the death registration States, Pub. Health Rep. 61, 259-265 (1946). II. The birth rate and infant and maternal mortality, Pub. Health Rep. 61, 1529-38 (1946).
- (2) United States Bureau of the Census: Vital Statistics Rates in the United States, 1900-40 (1943).

adjusted for age were then obtained by multiplying the crude rate by the ratio of the adjusted to the crude. The adjusted rate for 1844 by the rate for 1844 by the ratio of the crude annual rates as computed by the Bureau of the Census (2), and averages of the annual rates adjusted for age as described above. Rates shown on the charts are averages of the crude annual rates as computed by the Bureau of the Census (2), and averages of the annual rates as computed by the Bureau of the Census (2), and averages of the annual rates as described above.

(3) United States Bureau of the Census: Age-adjusted death rates in the United States, 1900-40: Iwao M. Moriyama. Vital Statistics—Special Reports, vol. 23, no. 1 (1945).

DEATHS DURING WEEK ENDED JANUARY 17, 1948

[From the Weekly Mortality Index, issued by the National Office of Vital Statistics]

Week ended	Corresponding week,
Jan. 17, 1948	1947
10, 150	9, 960
9, 960	30, 807
31, 881	846
671	2, 523
658	67, 232, 072
2, 219	14, 888
66, 858, 967	11. 5
14, 551	9, 4
	10, 150 9, 960 31, 881 671 658 2, 219 66, 858, 967 14, 551

Q FEVER STUDIES IN SOUTHERN CALIFORNIA

I. Recovery of Rickettsia burneti from raw milk 1 2

By R. J. Huebner, Senior Assistant Surgeon, W. L. Jellison, Parisitologist, M. D. Beck, R. R. Parker, Director, and C. C. Shepard, Surgeon

A previous report (1) of observations made during the spring of 1947 on the occurrence of 17 cases of Q fever in Los Angeles County and subsequent studies (2) of 100 additional cases, indicated that Q fever is endemic 6 in Southern California. Proximity to dairies by reason of occupation or residence was a common factor in the histories of more than 50 percent of the cases. Except for dairy workers, it was noted that the infected persons rarely used milk from nearby dairies. It was also found in fairly extensive serological surveys that 10 to 20 percent of the dairy cows in the Los Angeles area possessed serum antibodies for Q fever.

These studies, when completed, will be reported later. The purpose of this paper is to report the recovery of *R. burneti*, the causative agent of Q fever, from the raw milk of four widely separated dairies in Los Angeles County.

METHODS OF STUDY

Epidemiological data pointed to certain dairies as being involved in recent human cases (dairy workers or nearby residents). Raw milk from suspected dairies was tested as being the possible source of infection. The cows were prepared for milking in the usual manner by washing the udder with water, but the udders of some were further washed with 70 percent alcohol before specimens were taken. Specimens from individual cows were hand milked into sterile wide-mouth vials which were sealed immediately after sampling all four quarters of the udder. In some instances the specimens represented strippings taken after milking machines had been used.

Specimens of pooled milk were obtained in three ways: (1) By pooling at the Q Fever Laboratory samples obtained by the above method, (2) by taking samples from milk cans by means of milk dippers, and (3) by similarly collecting samples of milk which had been mixed in the pasteurizing vat of a bottling plant.

The fresh raw milk specimens in 3 to 5 cc. amounts were promptly injected intraperitoneally or subcutaneously into adult guinea pigs at the Q Fever Laboratory, or were frozen with carbon dioxide and

¹ This work has been facilitated by the Q Fever Laboratory, which was established September 12, 1947, in the endemic area of Southern California, as a cooperative undertaking of the National Institute of Health, the California State Department of Public Health, and the Los Angeles County Health Department.

³ From the Division of Infectious Diseases, NIH, Bethesda, Md.

³ From NIH, U. S. Public Health Service, Bethesda, Md.

⁴ From the Rocky Mountain Laboratory, U. S. Public Health Service.

⁸ From the California State Department of Public Health.

⁶ Cases have been recognized in Los Angeles, Ventura, Santa Barbara, and Orange Counties.

shipped to the National Institute of Health, Bethesda, Md., and the Rocky Mountain Laboratory of the National Institute of Health, Hamilton, Mont. At the Q Fever Laboratory, uninoculated control guinea pigs were kept in the same cages with milk-inoculated guinea pigs. One of each group of guinea pigs, showing elevated temperatures for 2 or 3 days, was sacrificed and the whole blood or spleen passed to other guinea pigs. All surviving guinea pigs, including the controls, were bled 30 to 35 days after inoculation, and the serums were tested by the complement fixation test 78 for Q fever antibodies. The development of antibodies in the serums of guinea pigs was regarded as evidence that the material inoculated was infected. However, in those instances where a disease-producing agent appeared to have been recovered, passages in guinea pigs were continued, and where strains of rickettsiae appeared to be well established, crossimmunity tests were performed with known strains of Q fever rickettsiae. Blood, spleen, or other tissues of well-established strains were inoculated into mice and into the yolk sacs of fertile hen's eggs. Yolk sacs which showed infection with a rickettsia-like agent were prepared as antigens and tested by the complement fixation test with standard Q fever serums, human and bovine serums from California, and control serums.

RESULTS

Rickettsial organisms, identified by all available criteria as R. burneti, were recovered by each of the three laboratories from the milk of four widely separated dairies located in Los Angeles County. A total of 50 milk specimens was injected into guinea pigs, 40 of them giving evidence of infection with R. burneti. Table 1 shows in some detail the isolation studies that have been completed on milk specimens from the four dairies as tested in two laboratories. Table 2 gives the results obtained in the third laboratory.

Dairy No. 1.—Approximately 12.5 percent of 1,050 cows from dairy No. 1 were found to possess antibodies for Q fever in the complement fixation test, and 28 cases of Q fever were found in persons living or working in close proximity to this dairy. The pooled milk of each of 33 milking strings of representing 28 to 30 cows was collected by methods described above. The 33 milk specimens were promptly frozen and shipped by air express to the National Institute of Health where they were inoculated into guinea pigs.

⁷ Both Bengtson and modified Kolmer techniques were used.

⁸ Henzerling and Nine Mile strains were used as antigen.

A group of cows milked together as a unit.

¹⁰ The milk specimens were cultured on blood agar plates and ascertained to be free of pathogenic bacteria detectable by that technique. We are indebted to Dr. Charles W. Bonynge of the University of Southern California for these tests.

Table 1.—Data on rickettsial agents recovered from raw milk of 4 dairies in the Los Angeles area as acquired by means of the complement fixation test, yolk sac cultivation and cross-immunity tests in the Q Fever Laboratory, Los Angeles County, and the National Institute of Health

Source of raw milk	Results of con ation test f serums of injected w specified la	mplement fix- or Q fever on guinea pigs ith milk at boratories	Rickettsial trains culti- vated in yolk sacs of fertile eggs (Nation- al Institute	Cross-immunity test with Dyer strain of R. burneti (National Insti-
	Q Fever Laboratory	National Institute of Health	al Institute of Health)	ional Insti- tute of Health)
Dairy No. 1:	*			
Dairy No. 1: Pool No. 1 (5 cows) Pool No. 2 (500 cows) Pool No. 3 (500 cows) Strings 28–30 cows each: 1	x x x	X		***************************************
strings 28–30 cows each: 1		x		*
2		x	· X	
3		_		
4		x		
5		x		
6		x		x
7		X	***********	
9		X X	**********	
10		x		
11		x		
12		x		
13				
14	**********	x	X	
15		X		
16	**********	x		×
18		x		
19	**********	-1	X 8	
20		x	x	
21	*********	x		X
22	******	X		
23	*******	x	**********	*******
24 25	**********	X X	X	X
26	******	x	A	A
27		x	X	x
28		x		
29		X -		
30		x	**********	
31		x		
32		x		
33 34		x x	x	*******
ndividual cows with mastitis of unknown	**********		^	
cause: 5203	_	_		
5708	_	_		
5282	-	-	*********	
7111	-	-	*********	*********
lows:				
7334		x	X X	
Dairy No. 2:				
Pool No. 1 (15 cows)	X			
Pool No. 1 (15 cows) Pool No. 2 (15 cows) ndividual cows:	_			
95	x			
129	_			
185	x			
Pairy No. 3: Pool No. 1 (10 cows) (serologically positive) Pairy No. 4:	x	x	**********	********
Pool No. 1 (30 cows) Pool of entire herd (90 cows)	X			

Note;

x = Positive.

- = Negative.

! = Group of cows milked together as a unit.

! = A rickettsia-like organism immunologically distinct from Q fever.

After an average incubation period of 9.7 days (variation 5 to 17 days) 26 of these specimens produced febrile episodes in guinea pigs. Twenty-nine specimens produced serum antibodies for Q fever in the guinea pigs which were bled 30 days after inoculation. Seven of the milk specimens therefore failed to produce fever, but only 4 failed to produce Q fever antibodies.

Guinea pigs which became febrile following inoculation with 8 of the specimens were sacrificed, and illnesses typical of the reaction following injection with Q fever rickettsiae were produced in other guinea pigs inoculated with the blood or spleen of these animals. Gross pathological changes typical of this infection were observed in second passage guinea pigs, i. e., large friable spleens and subcutaneous indurated nonsuppurative inflammatory reactions. Attempts to culture an agent by ordinary bacteriological methods from the blood and spleen of infected guinea pigs were negative.

Eight strains of rickettsiae were established in the yolk sacs of fertile hen's eggs by the use of infected guinea pig blood and tissues. The cultural, morphological, and tinctorial characteristics of these strains could not be distinguished from known strains of *R. burneti*. Antigens prepared from infected yolk sacs reacted specifically in the complement fixation test with standard Q fever serums and positive human and cattle serums from California. Positive reactions were not obtained with normal serums nor with serums positive for other diseases.

Complete cross immunity in guinea pigs which had been inoculated with the Dyer strain of Q fever rickettsiae was demonstrated with five guinea-pig California adapted strains.

Specimens of milk from each of 86 cows from dairy No. 1 were also injected into guinea pigs at the National Institute of Health. Complete results are not available. However, 13 of the 86 specimens produced antibodies in guinea pigs and 2 strains (6,832 and 7,334) were readily cultured in the yolk sacs of fertile hen's eggs.

Another organism fulfilling the cultural and tinctorial requirements of a rickettsia when grown on the yolk sac of fertile eggs was recovered from the pooled milk of string 19 (table 1), 1 of the 4 strings from which R. burneti was not recovered. This organism produced early onset of fever, ecchymosis, and necrosis of the scrotum and frequently death when it was inoculated into male guinea pigs. It was found in cross immunity and complement fixation tests to be immunologically distinct from R. burneti. Attempts to grow this organism on cell-free media have failed. This organism as well as an apparently identical organism recovered from the feces of cows represented in milk pool No. 1 (table 1) will be studied further.

At the Q Fever Laboratory, samples from 3 pools of milk from dairy No. 1 were inoculated into guinea pigs. Each of 2 speci-

mens represented pooled milk from half the cows of the dairy, approximately 500 cows each. Strains of Q fever rickettsiae were established in guinea pigs from each specimen. The third specimen represented the pooled milk of 5 cows which were serologically positive for Q fever. This specimen was divided and injected into guinea pigs at the National Institute of Health, as well as at the Q Fever Laboratory. Illnesses typical of Q fever and specific Q fever antibodies were produced in guinea pigs at both laboratories.

At the Rocky Mountain Laboratory, samples from 10 pools of milk representing 16 strings from dairy No. 1 were injected into guinea pigs. Nine recoveries of a rickettsial agent were made. One strain was cultivated in the yolk sacs of fertile hen's eggs and 9 strains were shown to produce immunity in guinea pigs to the Nine Mile strain of Q fever. These tests fully confirmed the results at the other 2 laboratories and indicated again that the recovered organisms were strains of R. burneti (table 2).

Table 2.—Complement fixation and immunity test on guinea pigs injected at the Rocky Mountain Laboratory with milk specimens from dairy No. 1

Strings ¹ of dairy cows from which pooled milk specimens were injected into guinea pigs	Results of complement fixation tests for Q fever on serums of guinea pigs injected with milk	Strains cul- tivated in yolk sac	Immunity to Nine Mile strain of R. burneti
String No.:			
1	X		x
5	X	**********	X
8	***********		X
Dayled atalanas	***********		X
Pooled strings: 12 and 24			
12 and 24 15 and 20	, A		χ.
21 and 22			x
23 and 26			x
28 and 30		X	x
19 and 33			x

 $^{^{\}rm 1}$ Pooled milk specimens taken from milking strings consisting of 28 to 30 cows identical with those listed in table 1.

Dairy No. 2.—A small dairy milking less than 100 cows was studied because a son of the dairy owner had recently contracted Q fever. Five specimens of milk were tested and results on 3 indicated infection with Q fever. One of the cows found to be shedding R. burneti was a young Guernsey, producing milk with a high butterfat content. No illness was apparent and the cow was regarded by the owner as one of his best producers.

Dairy No. 3.—Dairy No. 3, a dairy milking approximately 200 cows, was one of the dairies found during the preliminary studies in the spring of 1947 to have a number of serologically positive cows. One pool of milk representing 10 cows found to be positive by the complement fixation test was divided and injected into guinea pigs

Note.—x = Positive for Q fever. -= Negative for Q fever.

in both the Q Fever Laboratory and at the National Institute of Health. Organisms identical with R. burneti were recovered.

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Dairy No. 4.—Approximately one-third of the cows on dairy No. 4 (90 cows) were found to be positive for Q fever in the complement fixation test. A pool of milk representing the entire herd was tested at the National Institute of Health and at the Q Fever Laboratory. All injected guinea pigs developed fever after a relatively short incubation period and were subsequently shown to have antibodies for Q fever. Another pool of milk representing one string of approximately 28 cows inoculated at the Q Fever Laboratory likewise resulted in the production of Q fever antibodies in the guinea pigs and the establishment of a strain.

The raw milk of a fifth dairy was also studied. A single specimen of milk pooled from the entire herd of 130 cows was obtained at a bottling plant. Whole milk, cream, and resuspended sediment (concentrated 10 times) were each injected into guinea pigs. Studies which were incomplete at the time of writing this report indicated that *R. burneti* was not recovered.

No perceptible evidence of illness was apparent in cows which were found to be shedding *R. burneti* in milk. This observation was supported by the observations of five or more well qualified veterinarians. Several cows with demonstrable mastitis were tested. In each instance both blood and milk from the cows gave negative results for Q fever when injected into guinea pigs (table 1, numbers 5203, 5708, 5282, 711, and 129).

TESTS OF MATERIALS OTHER THAN MILK

Cattle blood.—Injections of whole blood and blood clots from more than 150 cattle, most of them lactating cows (some serologically positive) from dairies where there were human cases of Q fever, have not resulted in the recovery of R. burneti. However, most of these specimens were shipped unfrozen to the National Institute of Health and preserved at icebox temperatures for as long as a month before they were inoculated.

Urine and feces.—A pool of urine and a pool of feces taken from the cows represented in pool No. 1 from dairy No. 1 (table 1) were inoculated into guinea pigs. Q fever rickettsiae were not recovered from these excretions despite the fact that organisms were recovered from milk taken from the same cows at the same time. Other specimens of urine and feces have been tested with negative results; however, more extensive studies with these excretions are planned.

Sick calves.—Four blood specimens and one spleen specimen from calves ill with fever and diarrhea of undetermined origin were tested. Inoculation experiments and serological tests of recovered calves indicated that these animals were not infected with Q fever organisms.

Insects and arthropods.—Pooled specimens of flies,¹¹ mosquitoes,¹² and several species of free living mites,¹³ collected from alfalfa feed were injected into guinea pigs on a limited scale. Completely negative results were obtained. Spinose ear ticks ¹⁴ also were injected but tests are not yet complete.

VALIDITY OF RESULTS

Since the Q Fever Laboratory was located in an area in which this disease appeared to be highly endemic in both human and animal populations and since spontaneous infection of guinea pigs with this agent is known to occur in experimental laboratories (3), it was necessary to determine the likelihood of spontaneous infections in milkinoculated guinea pigs. The results of the inoculation experiments and a serological check of 20 normal guinea pigs and a large pool of guinea-pig serums (complement) indicated that guinea pigs raised commercially in the area were free of Q fever infection.

Fifty guinea pigs inoculated with materials other than milk and 20 uninoculated guinea pigs which were kept in cages with inoculated animals at no time showed signs of illness suggestive of infection with Q fever rickettsiae. These animals when bled were without exception negative for Q fever by the complement fixation test. In contrast to these negative results, 9 recoveries of this organism were made from

15 specimens of milk inoculated during the same period.

At the National Institute of Health the milk specimens reported on in this paper represented the first inoculations of experimental material to be made in a newly constructed laboratory. No instance of spontaneous Q fever infection in guinea pigs has been encountered

in this building to date.

The results obtained at the Rocky Mountain Laboratory were fully confirmatory of the results obtained in the other laboratories. Further evidence bearing on the validity of the results was provided by the fact that in seven instances where a pool of milk produced the infection in one laboratory, it was also found positive in another.

DISCUSSION

The relative ease with which R. burneti was recovered from milk of dairies in Los Angeles City and County suggests a high degree of availability of this pathogenic agent to the human and animal populations of the area, since nearly all of this milk is transported about the county before processing and much of it is sold raw. The occurrence

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¹¹ Siphona irritans (L.) and Musca domestica L.

¹² Cules quinquefasciatus Say.

¹⁸ Gohiera fusca Oudms and Histiostoma Sp.

¹⁴ Otobius megnini (Duges).

Note.-Footnotes 12, 13, and 14 determined at U. S. National Museum.

of Q-fever infection in the human population and a demonstrable widely disseminated source of *R. burneti* in the same area suggest a causal relationship. Whether or not milk represents an effective source of infection to man, however, cannot be determined by the data presented in this report.

The evidence presented by outbreaks in packing houses, stockyards, (4, 5) and laboratories (3) did not indicate that the drinking of infected milk was a cause of those outbreaks. A pulmonary route of infection was considered the most likely possibility in several of these outbreaks. Incomplete studies in California (1, 2) suggest that, for certain specific occupational and residential groups, the drinking of infected milk is an improbable mode of infection. However, the evidence did not rule out infected milk as a potential source of infection to man by some mode yet to be determined.

The failure to recover R. burneti from whole blood, blood clots, urine, and feces of a limited number of cows shedding R. burneti in their milk and the absence of a demonstrable illness in the infected animals suggested that a local infection of the udder may occur in the absence of concurrent severe systemic infection in the cow. The presence of R. burneti was not associated, however, with observable pathology in the udder or with diminution in either quantity or quality of milk.

SUMMARY

R. burneti, the causative agent for Q fever was recovered from the raw milk of four dairies in Southern California. These recoveries were made in three laboratories: The Q Fever Laboratory, Los Angeles County, Calif.; The National Institute of Health, Bethesda, Md.; and the Rocky Mountain Laboratory, Hamilton, Mont. Seven isolations were made from duplicate specimens studied in two of the three laboratories.

The isolation of R. burneti from milk was established on the basis of the following manifestations of the recovered strains:

1. Febrile episodes typical of Q fever were produced in guinea pigs, and gross pathological findings typical of infection with Q fever rickettsiae were observed.

2. Specific antibodies for Q fever were demonstrated in the serums of guinea pigs previously injected with milk. The failure of such antibodies to appear in a large group of uninoculated control guinea pigs and the guinea pigs injected with materials other than milk provided good evidence against the occurrence of spontaneous infection in the guinea pig colonies.

3. Cross-immunity tests performed at the National Institute of Health and at the Rocky Mountain Laboratory showed that five newly isolated strains were identical with the Dyer strain of R. burneti and nine strains were shown to produce

immunity to the Nine Mile strain of R. burneti.

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4. Rickettsia-like organisms were cultivated in the yolk sac of fertile hen's eggs from the blood and spleen of guinea pigs inoculated with milk. The cultural, morphological, and tinctorial characteristics of eight such strains were identical with those of *R. burneti*.

5. Yolk sac antigens prepared from milk strains by the usual techniques reacted specifically in the complement fixation test with standard Q fever serums.

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6. Serums from California cows and humans found to contain antibodies for Q fever were tested with antigens prepared from milk strains. Specific reactions occurred in the complement fixation test.

While R. burneti was recovered from raw milk the available epidemiological evidence did not indicate that the drinking of milk was the cause of the majority of cases which have been studied thus far. However, that infected milk may serve as a source of infection to man by some mode as yet undetermined appeared to be a distinct possibility.

ADDENDUM

Tests. 15 as yet incomplete, indicate that the two methods of pasteurization in general use in two large commercial milk plants rendered the raw milk naturally infected with R. burneti apparently noninfectious for guinea pigs. Three tests using the vat method and four tests using the high-temperature short-time method of pasteurization have been completed. A fourth experiment with vat pasteurization was incomplete at the time of preparation of this manuscript.

ACKNOWLEDGMENTS

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We are indebted to Dr. David L. Lackman for the serological testing of many specimens and to Lt. Col. Arthur Long, Surgeon General's Office, A. U. S., for laboratory assistance in the portion of this work performed at the National Institute of Health.

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¹⁸ Report, Feb. 9, 1948, from the Q Fever Laboratory to the Director, California State Department of Public Health.

TWO NEW SALMONELLA TYPES: SALMONELLA HIDALGO AND SALMONELLA MISSION 1

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By James Watt, Surgeon, and Thelma M. DeCapito, Assistant Bacteriologist, United States Public Health Service, P. R. Edwards and Alice B. Moran, National Salmonella Center 1

Two new Salmonella types have been isolated in the course of diarrheal disease studies in Hidalgo County, Texas.

A. Organisms of the Salmonella group are known to infect a large number of animals. In the present investigation of the epidemiology of salmonellosis in humans, routine cultures are being made on a series of domestic animals in the study areas. The organism described below as Salmonella hidalgo was isolated from a duck that was examined as a part of this work.

The specimens are collected by inserting a cotton-tipped applicator into the rectum or cloaca of the animal or fowl being studied. The entire swab is then placed in a tube of tetrathionate broth (Difco) which is incubated for 20–24 hours and then plated on SS agar (Difco). Suspicious colonies are fished to Kligler's iron agar and then identified in the routine manner. Specimens from a dog, a cow, and three ducks were cultured at the home involved. The cultures from the dog, cow, and one duck were negative. The culture from the second duck was positive for Salmonella anatum, and Salmonella hidalgo was isolated from the third duck culture.

The complete description of the organism follows: S. hidalgo possessed the cultural and biochemical characteristics generally attributed to the Salmonella group, except that it produced a slight acidity in salicin broth after 33 days' incubation. Hydrogen sulfide was produced, but indol was not formed nor was gelatin liquefied. Acid and gas were produced from glucose, arabinose, maltose, trehalose, rhamnose, xylose, dulcitol, sorbitol, and mannitol within 24 hours. Cellobiose was fermented after 5 days. Lactose, sucrose, raffinose, and inositol were not attacked. Jordan's tartrate was acidified.

On serological examination the organism was strongly agglutinated by S. newport O serum (VI, VIII) and in absorption tests removed all agglutinins from that serum. Examination of the H antigens revealed that the organism was diphasic. Phase 1 was agglutinated to the titer of S. rubislaw phase 1 serum (r) and completely removed H agglutinins from the serum in absorption tests. Phase 2 was agglutinated strongly by S. abortus-equi serum (enx) and by S. glostrup,

¹ From the Division of Infectious Diseases, National Institute of Health, Pharr, Texas, and the Department of Animal Pathology, Kentucky Agricultural Experiment Station, Lexington, Kentucky. The work reported here was done in part in connection with a project of the Kentucky Agricultural Experiment Station and is published by permission of the Director. This portion of the work was supported by a research grant from the United States Public Health Service

phase 2 serum (enz₁₅). Absorption tests showed that phase 2 of S. hidalgo was identical with phase 2 of S. glostrup. The antigenic formula of S. hidalgo is VI, VIII: r-enz₁₅.

B. A previous communication described a new Salmonella type (1) isolated from humans in Hidalgo County, Texas. Salmonella mission, another new type was isolated in October 1946 in a continuation of these studies.

This organism was isolated from an SS agar plate prepared by streaking with a rectal swab. The organism was not recovered from tetrathionate broth in which the swab was incubated.

The patient was an 18-month-old Spanish-American male. The child had not had any diarrheal or other disease during the 6 months he had been followed prior to October 14, 1946, nor did he develop any illness during the next 6 weeks. Cultures were made in April, May, June, July, and August, 1946, and no Salmonellas were isolated from any of these cultures. No culture was made in September as the child was in Mexico from September 14 to 25. Again, in November cultures were made, and no Salmonellas were isolated. A specimen from a 5-year-old sibling was obtained at the same time and was found to be negative.

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A description of the organism follows: The biochemical properties of S. mission were the same as those given for S. hidalgo except that it did not ferment salicin and that gelatin was liquefied after 65 days incubation.

S. mission was agglutinated strongly by S. oranienburg O serum (VI, VII) and in absorption tests removed all agglutinins from the serum. The culture was diphasic and phase 1 was agglutinated to the titer of S. typhi H serum (d). In absorptions it reduced the titer of S. typhi serum from 1-10,000 to 1-200. It completely removed agglutinins for phase 1 of S. oregon, S. muenchen, and S. stanley from the serum. Phase 2 was agglutinated by serums for all the nonspecific phases of the Kauffmann-White classification. When tested with absorbed serums for factors 2, 3, 5, 6, 7, 10, and 11 it was agglutinated only by factor 5 serum. In absorption tests it was found that phase 2 of S. mission was not identical with phase 2 of S. thompson. Lack of uniformity in the 1-5 phases long has been recognized.

The diagnostic formula for S. mission is VI, VII: d-1, 5.

A second strain of S. mission was isolated from a rectal swab culture of a cat in April 1947: This specimen, cultured in the manner described in A above, was obtained in a town 15 miles from the original source.

REFERENCE

Watt J.; DeCapito T.; and Moran, A. B.: A new Salmonella type isolated from man: Salmonella texas. Pub. Health Rep., 62: 808-809 (May 30, 1947).

INCIDENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

REPORTS FROM STATES FOR WEEK ENDED JANUARY 24, 1948

Summary

A slight increase in the incidence of influenza was reported, from 10,360 cases to 11,687 for the current week, as compared with 4,129 for the corresponding week last year and a 5-year (1943-47) median of 4,387. Of the current total, 10,696 cases (92 percent) occurred in 8 South Atlantic, South Central, Mountain, and Pacific States, as follows (last week's figures in parentheses): Virginia 949 (868), South Carolina 1,218 (880), Tennessee 233 (110), Alabama 344 (265), Arkansas 586 (439), Texas 5,027 (4,509), Arizona 1,274 (1,039), California 1,065 (1,023). Only 3 other States reported more than 76 cases—Wisconsin 108 (last week 51), West Virginia 139 (last week 159), and Oklahoma 161 (last week 442). The cumulative total for the first 3 weeks of the year is 32,382, as compared with 12,522 and 239,498, respectively, for the same periods of 1947 and 1944, and a 5-year median of 12,712.

Of 46 cases of poliomyelitis reported for the week (last week 40, same week last year 69, 5-year median 27), Idaho reported 10, New York 5, and North Carolina 4. For the first 3 weeks of the year 127 cases have been reported, as compared with 239 for the corresponding period last year and a 5-year median of 111.

One case of smallpox was reported, in Louisiana. Massachusetts and Pennsylvania each reported 1 case of anthrax. Totals reported for the first 3 weeks of the year for certain other diseases (corresponding week last year and 5-year medians in parentheses) are as follows: Diphtheria 729 (988, 1,014); the dysenteries, combined, 2,271 (1,834, 1,610); infectious encephalitis 14 (20, 22), measles 23,405 (10,949, 13,573); meningococcus meningitis 263 (266, 711); scarlet fever 6,330 (6,844, 10,749); smallpox 9 (13, 30); tularemia 74 (154, 87); typhoid and paratyphoid fever 111 (127, 132); endemic typhus fever 56 (155, 191), whooping cough 7,321 (6,582, 6,526).

Deaths recorded during the week in 93 large cities of the United States totaled 10,244, as compared with 10,150 last week, 9,958 and 10,157, respectively, for the corresponding weeks of 1947 and 1946, and a 3-year (1945-47) median of 9,958. The total for the 4 weeks ended January 24 is 42,125, as compared with 40,765 for the same period in 1947. Infant deaths during the week totaled 722, as compared with 671 last week, and a 3-year median of 622. The cumulative figure is 2,940, as compared with 3,378 same period last year.

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In these tables a zero indicates a definite report, while leaders imply that, although none was reported, cases may have occurred.

	D	iphthe	ria	1	Influenz	8		Measles	3		eningi ingoco	
Division and State	Wende	eek ed—	Me-	Wend	eek ed—	Me-	w	eek ed—	Me-	wende	eek ed-	Me-
	Jan. 24, 1948	Jan. 18, 1947	dian 1943- 47	Jan. 24, 1948	Jan. 18, 1947	dian 1943- 47	Jan. 24, 1948	Jan. 18, 1947	dian 1943- 47	Jan. 24, 1948	Jan. 18, 1947	dian 1943- 47
NEW ENGLAND												
Maine	1 0	2	1 0	1	3 2	3	3 2	190	29	1 0	0	2
New Hampshire Vermont	0	0	0		42	42		179	28	1	0	
Massachusetts	5	10					388	431	409	2	4	8
Rhode Island Connecticut	0	0	0 2	4	2	8	16	215	17 65	2	0 2	1
MIDDLE ATLANTIC							-					
New York	11	21	18	12	1 13	1 15		209	573	5	11	27
New Jersey Pennsylvania	13 11	13	10	(1) 2	14	18	543 391	85 640	85 656	6	6	12
BAST NORTH CENTRAL	11	10	10	(-)		- 4	301	010	000			**
Ohio	10	9	9	12	7	18	560	330	82	4	2	8
Inglana	10	5	5	13	3	16	249	24	61	1 3	1	7
Illinois Michigan 3	12	15	4 15	8	2	11	1, 483 719	35 46	177 129	1	2	13
Wisconsin	5	3	3	108	46	101	275	71	71	3	3	8
WEST NORTH CENTRAL		-										
Minnesota	2	6	6		1		385 312	30 10	16 95	0 5	- 2	3
Missouri	8	1	4	15	4	12	39	2	45	0	4	1 8
North Dakota	0	0	1	1	34	34	93 20	1	33	1	0	10
South Dakota Nebraska		0	0	40	13	51	7	16 14	13	1	1	2
Kansas	2 3	14	3	76	67	67	9	9	130	1	0	5
SOUTH ATLANTIC												
Delaware Maryland 3	6	15	12	3	5	26	61 30	158	33	2 3	0 2	0
District of Columbia.	0	0	0			3	63	21	17	0	0	11
Virginia West Virginia	10	10	10	949 139	596 51	763	136 244	67	116 18	2	1	11
North Carolina	17	7	12		01	51	2	169	59	3	20	7
South Carolina	15	1 9	6	1, 218	713	775	24	46	46	1	1	1
GeorgiaFlorida	8	6	7	62 18	14 20	66 13	27 44	150	41 21	0	0	3
EAST SOUTH CENTRAL						-						
Kentucky	5	13	6	5	2	16	11	2	25	1	1	5
Tennessee	5	6 5	6	233 344	39 50	78 175	98	35 8	48	1 2	1 2	6
Miasissippi 3	2	5	5	48			30			ō	1	3
WEST SOUTH CENTRAL												
Arkansas	2	9	10 6	586	105 35	148	85 267	58	52 11	0	4	5
LouisianaOklahoma	11	0	5	161	114	35 126	6	6	19	1	0	3
1 0xa3	19	26	44	5, 027	1, 788	2, 094	778	71	111	12	6	10
MOUNTAIN							07	105				
Montana Idaho	3	0	0	31 32	30	35	97 10	135	54	0	0	0
W yoming	0	2	0		6	6	122	7	10	0	1	1
Colorado New Mexico	6	6	4	50 6	15	57	69	25 13	109	0	0	0
Arizona	4	4	4	1, 274	259	259	12	43	14	0	0	0
Utah 3	0	0	0	55	5	7	11	8	32	0	1	0
Nevada	0	U	0						*		0	0
Washington	2	11	8	31 _		1	102	19	140	3	0	2
Oregon California	2 7	4	4	58	14	33	33	25	71	0	2	4
Total	252	282	312	1, 065	4, 129	4, 387	8, 797	3, 739	5, 490	86	83	240
	729										266	-
weeks			1, 014	32. 382		12, 712		10, 949	-	263		711
Seasonal low week 4.		July 5 8, 554		(30th) J1 75, 940			_	33, 836		(37th)	sept. 1	3-19

New York City only.
 Philadelphia only.
 Period ended earlier than Saturday.
 Dates between which the approximate low week ends. The specific date will vary from year to year.

Telegraphic morbidity reports from State health officers for the week ended Jan. 24, 1948, and comparison with corresponding week of 1947 and 5-year median—Con.

litis		Sci	arlet fer	ver	8	mallpo	X	Typh	old and hold fe	l para ver
Me-		We		Me-	We	ek ed-	Me-	wend	eek ed-	Me-
dian 1943- 47	- J	an. 24, 948	Jan. 18, 1947	dian 1943– 47	Jan. 24, 1948	Jan. 18, 1947	dian 1943- 47	Jan. 24,5 1948	Jan. 18, 1947	dian 1943- 47
0		13	35 8	35 8	0	0	0	0	0	0
1	1	17	7	7	0	0	0	0	1	0
0		86	172 14	287 14	ő	0	0	0	0	1
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			000	270					6	
4	1	244 102	290 104	372 107	0	0	0	2	1	1
0		184	147	285	0	0	0	3	1	3
0		287 49	287 83	287 100	0	1 2	1 2	0	1 0	1
1	1	152	126	221	0	0	0	0	0	i
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0		9	25	12	0	0	0	0	0	0
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0	0	15	18	18	0	0	0	3	0	2
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			1	-						
1		4	4	6	0	0	0	0	0	0
0		6 22	5	11 25	1	0	0	0	4	1
3	3	36	40	103	0	Ö	0	8	5	5
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0		23	11 13	14 14	0	0	0	0	1	1 0
0	0	8	6	6	0	0	0	0	0	0
0	0	22	53	53 10	0	0	0	0	0	1
0		11	14	11	0	0	0	1	0	1 1 2 0
0	1	36	28	45	0	0	0	0	0	0
9	4	9	٩	4	4	ď	1	٩	9	U
2	2	57	. 30	57 24	0	0	0	0	0	0
0	0	34 81	13 106	24 206	0	0	0	8	0	0 2
	-				1	5	13	40		51
		_		-					-	132
-	-	-	1		1	T			-	4, 708
	11 15 –21	111 6, 15 -21 (3	111 6, 353 15 -21 (32nd)	111 6, 353 6, 844	111 6, 353 6, 844 10, 749 15 -21 (32nd) Aug. 9-15	111 6, 353 6, 844 10, 749 9 15 -21 (32nd) Aug. 9-15 (35th) A	111 6, 353 6, 844 10, 749 9 13 15 -21 (32nd) Aug. 9-15 (35th) Aug. 30-8	111 6, 353 6, 844 10, 749 9 13 30 15 -21 (32nd) Aug. 9-15 (35th) Aug. 30-Sept. 8	111 6, 353 6, 844 10, 749 9 13 30 111 15 -21 (32nd) Aug. 9-15 (35th) Aug. 30-Sept. 5 (11th)	111 6, 353 6, 844 10, 749 9 13 30 111 127 15 -21 (32nd) Aug. 9-15 (35th) Aug. 30-Sept. 5 (11th) Mar.

n 3-

8 12

 ³ Period ended earlier than Saturday.
 ⁴ Dates between which the approximate low week ends. The specific date will vary from year to year.
 ⁴ Including paratyphoid fever reported separately as follows: Massachusetts 2 (salmonella infection);
 Georgia 1; Tennessee 1; Arizona 1; California 1.

Telegraphic morbidity reports from State health officers for the week ended Jan. 24, 1948, and comparison with corresponding week of 1947 and 5-year median—Con.

	Who	oping c	ough		V	Veek er	nded Ja	nuary 24	, 1948		
	Week e	ended-	Me-	D	ysente	ry	En-	Rocky Mt.		Ty-	Un-
Division and State	Jan. 24, 1948	Jan. 18, 1947	dian 1943- 47	Ame- bic	Bacil- lary	Un- speci- fled	ceph- alitis, infec- tious	spot- ted fever	Tula- remia	phus fever, en- demic	du- lant feve
NEW ENGLAND	90		10								
Maine New Hampshire	38	14	16								
Vermont	132	30	34								
Massachusetts	115	226	111		2						
Rhode Island Connecticut	28	45 59	27 47		1						
		- 1	-								
New York	129	251	251	12	10						1
New Jersey	88	139	139	8	1						
Pennsylvania	84	225	220				1	*******			
PAST NORTH CENTRAL	151	101	101								
Indiana	30	36	16								
Illinois	70	133	100	5	2				1		1
Michigan ⁸ Wisconsin	139 122	219 135	129 98	1	1	*****			1		
WEST NORTH CENTRAL	144	100	90						1		
Minnesota	44	9	35			1					
lows	5	7	10	2							
Missouri	30	17	6			2					
North Dakota	26	3	2			-					
Nebraska	2 10	2	5	1							
Cansas	73	19	26				1				
SOUTH ATLANTIC											
Delaware	3								2		
Maryland 3 District of Columbia	41 5	96	60			0				*****	
Jirginia	51	39	70	1		51			1	1	
Virginia	13		29								
North Carolina	59	23	119							2	
outh Carolina	158	39	53	3	2 2				····i	3	
Georgia	12 28	7 25	20	1						6	
EAST SOUTH CENTRAL											
Kentucky	7	48	33								
Cennessee	34 49	28 50	28 15	1		1	1				
Mabama	5	80	10	*****	1				1	*****	
WEST SOUTH CENTRAL	1				-						
rkansas	25	5	15	3							
ouisiana	10	7	3	1					1		
Oklahoma	39 387	11 252	10 193	18	280	66			1	3	
MOUNTAIN	357	202	193	18	200	00			1	9	
Montana	10	3	5	1							
daho	5	1	2 5								
Vyoming	10	1		1							
Colorado	59 12	1 3	30 11								
rizona	35	20	19			21			1		
Jtah 3	15		14						1		
levada			1								
PACIFIC	70	90	90								
Vashington	78 6	32 10	32 10	5							
California	97	112	123	2	4						
Total	2, 576	2, 485	2, 418	67	306	147	4	0	11	15	10
ame week: 1947	2, 485		= 1.0	22	344	67	===	0	60	56	-
Median, 1943-47	2, 418			27	309	67	8	0	35	56	8 7
weeks: 1948	2, 418 7, 321			167	1.047	1,057	14	2	75	56	26
1947	6, 582			77	1,093	664	20 22	1 0	154	155	25
Median, 1943-47	6, 526			81	1,093	436	22	0	87	191	6 20

Period ended earlier than Saturday.
 3-year median, 1945-47.

Anthrax: Massachusetts 1, Pennsylvania 1. Territory of Hawaii: Leprosy 1, paratyphoid fever 1, whooping cough 33.

WEEKLY REPORTS FROM CITIES 1

City reports for week ended Jan. 17, 1948

This table lists the reports from 87 cities of more than 10,000 population distributed throughout the United states, and represents a cross section of the current urban incidence of the diseases included in the table.

	cuses	ses	Influ	enza	28	me-	nia	litis	ever	38	and hoid	ongh
Division, State, and City	Diphtheria c	Encephalitis, in fectious, cases	Cases	Deaths	Measles cases	Moningitis, me- ningococcus, cases	Pneumo deaths	Poliom yelitis cases	Scarlet fev	Smallpox cases	Typhoid and paratyphoid fever cases	Whooping cough
NEW ENGLAND												
Maine: Portland	0	0		0		0	1	0	0	0	0	1:
New Hampshire: Concord	0	0		0		0	1	0	0	0	0	
Vermont:	0	0		0		0	1	0	0	0	0	1
Massachusetts:	4	0		1	99	0	23	0	29	0	0	1
Boston	0	0		0		0	0	0	0	0	0	1:
Fall River Springfield	0	0		0	1	0	0	0	7	0	0	
Worcester Rhode Island: Providence	0	0		0		0	2	0	5	0	0	
Connecticut: Bridgeport	0	0		0	2	0	0	0	7	0	0	
Hartford New Haven	0	0		0		0	2	0	3	0	0	
MIDDLE ATLANTIC												
New York:								١.	-			
Buffalo	1	0	3	0	355	1 5	5 75	0 2	70	0	0 2	1: 2
New York Rochester	11	1 0	3	4 0	2	0	3	1	12	0	0	
Syracuse New Jersey:	ő	0		ő	11	0	0	0	10	0	0	1
Camden	0	0		0	16	0	0	0	14	0	0	
Newark Trenton	0	0	1	0	3	0	4	0	3	0	0	
Pennsylvania: Philadelphia	3	0	4	2	57	1	37	0	48	0	1	3
PittsburghReading	0	0		0	1	0	5	0	9 8	0	0	1.
EAST NORTH CENTRAL												
Ohio:				1	13	0	11	0	18	0	0	
Cincinnati Columbus Indiana:	2	0	1 3	3	67	0	2	0	10	0	0	1
Fort Wayne	0	0		0	2 44	0	2 4	0	5 3	0	0	
Indianapolis South Bend	0	0		0	44	0	0	0	0	0	0	
Terre Haute	ő	0		0	18	0	1	0	1	0	0	****
Illinois: Chicago	1	0	2	0	383	2	25	0	52	0	0	2
Michigan: Detroit	3	0	1	0	23	1	19	1	66	0	0	2
Flint Grand Rapids	0	0		0	123	0	3	0	3	0	0	
Wisconsin:				0	16	0	. 0	0	0	0	0	
Kenosha Milwaukee	0	0		0	7	0	1	0	16	0	0	1
Racine	0	0		0	15	0	1 0	0	0	0	0	
Superior	0	0	*****	0	*****	0	0	0	0		1	1
WEST NORTH CENTRAL												
Minnesota Duluth	0	0		0	2	0	1	0	3	0	0	1
Minneapolis	0	0		0	148	0	4	0	15	0	0	2
St. Paul Missouri:	0	0		0	8	0	5	0	5			
Kansas City	0	0	2	0	3	2	1	0	3 2	0	0	1
St. Joseph	0	0	2	0	1 22	0	0	0	14	0	0	*****

¹ In some instances the figures include nonresident cases.

City reports for week ended Jan. 17, 1948—Continued

	cases	tis, in-	Influ	enza	90	me-	nis	litis	Ver	ses	hoid	ongh
Division, State, and City	Diphtheria o	Encephalitis, fectious, case	Cases	Deaths	Measles cases	Meningitis, meningococcus,	Pneumon deaths	Poliomyelitis cases	Scarlet fever	Smallpox cases	Typhoid and paratyphoid lever cases	Whooping cough
WEST NORTH CENTRAL— continued						-						
North Dakota Fargo	0	0		0	111	0	2	0	1	0	0	8
Nebraska:					3	0	4	0	5	0	0	3
Omaha Kansas:	0	0		0								
Topeka Wichita	0	0		. 0	2	0 1	3	0	3 4	0	0	6 5
SOUTH ATLANTIC												
Delaware: Wilmington	1	0		0	3	0	3	0	1	0	0	
Maryland: Baltimore							8	0	13	0	0	98
Baltimore Cumberland	2 4	0	3	0	3	0	1	0	1	0	0	35 1
District of Columbia:	0	0	1	1	49	2	9	0	10	0	0	11
Washington Virginia:			-		10						0	
Lynchburg	0	0		0		0	3 2	0	5	0	0	8
ROBHOKe	0	0		0		0	0	0	0	0	1	
West Virginia: Charleston	0	0		0	9	0	6	0	0	0	0	*****
Wheeling North Carolina:	0	0		0	1	0	3	0	2	0	0	*****
Raleigh	0	0		0		0	2	0	0	0	0	
Wilmington	1 0	0		0		0	4	0	0	0	0	. 1
South Carolina:	0	0	83	1	1	0	2	0	0	0	0	2
Charleston					•							-
Atlanta Brunswick	. 0	0	22	0		0	9	0	3	0	0	
Savannah	0	0	5	0	1	0	2	0	2	0	0	3
Florida: Tampa	1	0	1	0	16	0	4	0	2	0	0	8
EAST SOUTH CENTRAL								i				
Tennessee:							-		.			
Memphis Nashville	1 0	0	1	0	23	0	5 7	0	1 4	0	2 0	8
Alabama:	0	0	5	0	1	1	6	0	0	0	0	1
Birmingham Mobile	0	0	11	0		ō	2	ő	i	ő	0	
WEST SOUTH CENTRAL												
Arkansas:	0	0	4	0		0	1	0	1	0	0	1
Little RockLouisiana:												
New Orleans Shreveport	0	0	3	0	3	0	7 6	1 0	2 0	0	0	3
)klahoma:	0	0		0	54	1	4	0	2	0	0	4
Oklahoma City					94							
Galveston	0	0	1	0		0	6	0	0	0	8	4
Houston	1 0	0	5 3	0	15	0	7 1	0	1 0	0	0	10
San Antonio	0	0	0	*	1	0	*	0	١	0	۰	-
Montana:												
Billings	0	0		0	4	0	0	0	1	0	0	1
Great Falls	0	0		0	4	0	0	0	0	0	0	
Missoula	0	0		0		0	0	0	0	0	0	1
Denver	2	0	9	0	17	0	2	0	4	0	0	28 14
Pueblo	0	0		0		0	0	0	2	0	0	14
Salt Lake City	0	0		0	5	0	1	0	2	0	0 .	

City reports for week ended Jan. 17, 1948-Continued

Division, State, and City	cases	Encephalitis, in- fectious, cases	Influenza			me-	nia	litis	976	898	hold	cough
	Diphtheria		Cases	Deaths	Measles cases	Meningitis, me ningococcus cases	Pneumo deaths	Poliom ye cases	Searlet fe	Smallpox cases	Typhoid paratyp fever case	ng
PACIFIC												
Washington:					11		1	1 ,	9		0	
Seattle	0	0		0	1 1	0	1	1 1	i	0	0	
Tacoma	0	ő		ő	45	0	1 0	0	Ô	0	Ö	
California:	•	"						"	-			
Los Angeles	3	0	461	11	22	3 0	25 3 13	1	11	0	0	36
Sacramento	3 0 5	0	1	1	3	0	3	0	1	0	1	
San Francisco	5	0	15	0	151	1	13	1	13	0	0	- 0
Total	56	1	653	38	1,904	22	432	9	576	0	10	543
Corresponding week, 1947	102		84	16	894		449		558	0	4	747 600
A verage, 1943-47 1	75		1,065	1 49	31,874		2 491		970	0	9	600

1 Exclusive of Oklahoma City.

³ 3-year average, 1945-47. ³ 5-year median, 1943-47.

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Anthrax.—Cases: Philadelphia, 1.

Dysentery, amebic.—Cases: Philadelphia, 1; Atlanta, 1; New Orleans, 6; Los Angeles, 2; San Francisco, 1.

Dysentery, bacillary.—Cases: New York, 1; Memphis, 2; Los Angeles, 2.

Dysentery, unspecified.—Cases: Baltimore, 4; San Antonio, 1.

Leprosy.—Cases: Los Angeles 1.

Tularemia.—Cases: Baltimore, 1; Atlanta, 3; New Orleans, 2.

Typhus fever, endemic.—Cases: New York, 1; Kansas City, 3; Savannah, 1; Los Angeles, 1.

Rates (annual basis) per 100,000 population, by geographic groups, for the 87 cities in the preceding table (latest available estimated population, \$3,633,900)

	case	Encephalitis, in- fectious, case	Influenza		rates	me-	death	case	case	rates	para- ever	cough
	Diphtheria rates		Case rates	Death rates	Measles case	Meningitis, ningococcus, rates	Pneumonia d	Poliomyelitis rates	Scarlet fever	Smallpox case rates	Typhoid and typhoid for case rates	Whooping case rates
New England	10. 5 8. 3 4. 8 8. 0 14. 8 5. 9 7. 6 16. 5 12. 7	0. 0 0. 5 0. 0 0. 0 0. 0 0. 0 0. 0 0. 0	0. 0 3. 7 4. 8 8. 0 188. 8 100. 3 40. 6 74. 3 754. 4	2.6 4.2 2.7 2.0 6.6 0.0 17.8 0.0 19.0	267 206 487 398 136 148 185 248 368	0.0 3.2 2.1 8.0 3.3 5.9 2.5 0.0 6.3	112. 4 62. 0 47. 9 51. 7 96. 9 118. 0 86. 4 24. 8 68. 0	0.0 1.4 0.7 0.0 0.0 0.0 2.5 0.0 6.3	152 84 121 109 74 35 25 74 55	0. 0 0. 0 0. 0 0. 0 0. 0 0. 0 0. 0 0. 0	0.0 1.4 0.0 0.0 1.6 11.8 7.6 0.0 1.6	170 52 56 169 120 53 61 372 74
Total	8.7		101. 5	5. 9	296	3.4	67. 2	1.4	90	0.0	1.6	84

FOREIGN REPORTS

CANADA

Provinces—Communicable diseases—Week ended January 3, 1948.— During the week ended January 3, 1948, cases of certain communicable diseases were reported by the Dominion Bureau of Statistics of Canada as follows:

Disease	Prince Edward Island	Nova Scotia	New Bruns- wick	Que- bec	On- tario	Mani- toba	Sas- katch- ewan	Al- berta	British Colum- bia	Total
Chickenpox Diphtheria		13	7	26 6	452	47	31	32	74	682
Dysentery:					1				-	1
Unspecified Encephalitis, infectious					i		2	******	1	1 3
German measles				2	11			6	5	24 38
Influenza Measles		31		198	452	3	2	7	6 72	734
Meningitis, meningococ-					3	1		1		5
Mumps Poliomyelitis		29	2	74	328	26	19	37	26	541
Scarlet fever		2	3	16	98	3		4	10	136
Tuberculosis (all forms) Typhoid and paratyphoid		5	10	31	37	30	13		58	184
feverUndulant fever				1	1			1 2		3
Venereal-diseases:		******								
Gonorrhea	4	9	11 6	83 39	95 49	31	29	28 2	83 23	375 135
Other forms Whooping cough		3		20	20	18	2 2	18	17	98

REPORTS OF CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER RECEIVED DURING THE CURRENT WEEK

NOTE.—Except in cases of unusual incidence, only those places are included which had not previously reported any of the above-mentioned diseases, except yellow fever, during recent months. All reports of yellow fever are published currently.

A table showing the accumulated figures for these diseases for the year to date is published in the Public Health Reports for the last Friday in each month.

Smallpox

Iran.—For the week ended December 12, 1947, 38 cases of smallpox with 2 deaths were reported in Iran.

Siam (Thailand)—Bangkok.—For the period January 1-21, 1948, 24 cases of smallpox with no fatalities were reported in Bangkok, Siam.

Yellow Fever

Belgian Congo—Orientale Province—Uele District—Bondo.—Information dated January 16, 1948, stated that 1 fatal case of yellow fever was reported in Bondo, Uele District, Orientale Province, Belgian Congo.

FEDERAL SECURITY AGENCY

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UNITED STATES PUBLIC HEALTH SERVICE

THOMAS PARRAN, Surgeon General

DIVISION OF PUBLIC HEALTH METHODS

G. St. J. Perrott, Chief of Division

The Public Health Reports, first published in 1878 under authority of an act of Congress of April 29 of that year, is issued weekly by the United States Public Health Service through the Division of Public Health Methods, pursuant to the following authority of law: United States Code, title 42, sections 241, 245, 247; title 44, section 220.

It contains (1) current information regarding the incidence and geographic distribution of communicable diseases in the United States, insofar as data are obtainable, and of cholera, plague, smallpox, typhus fever, yellow fever, and other important communicable diseases throughout the world; (2) articles relating to the cause, prevention, and control of disease; (3) other pertinent information regarding sanitation and the conservation of the public health.

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